SATeLLITE CELLS: A REVIEW OF THEIR PHYSIOLOGY, MANIPULATION AND IMPORTANCE TO MUSCLE DEVELOPMENT

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Summary

Meat yield of a market age broiler is determined by the number of muscle fibre cells present at hatch. Muscle growth (hypertrophy) is influenced by the activity of satellite cells (located within the muscle fibre) that remain active for up to seven days post-hatch. Satellite cells respond to various stimuli (nutritional, environmental, physiological etc.) and thus potentially may be stimulated to increase meat yield and improve product uniformity. In particular, we are interested in increasing the yield of breast meat while being cognisant of associated potential for muscle myopathy.

I. INTRODUCTION

It has been demonstrated that muscle size (meat yield) is a direct function of muscle fibre cell number (as established by embryonic and post hatch (to seven days) satellite cell differentiation to breast muscle cells) and their subsequent growth (hypertrophy). Similarly, it is well known that breast yield from the modern day broiler chicken can vary from 17 to 25% of the birds' body weight; therefore, yield of breast meat (estimated to equate to 60% of the value of the whole bird) can dramatically influence profitability of the industry. Therefore it is important to increase the yield or breast muscle (and therefore value of bird) as well as achieve higher product uniformity. The objectives of this paper are to review the physiology of satellite cells and their function in relation to manipulation during the embryonic and post-hatch stages of development in the broiler chicken.

II. MUSCLE CELL PHYSIOLOGY

The muscle cell, in contrast to most other cells is multi-nucleated. These nuclei are found along the entire length of the muscle fibre with each nucleus exerting control over its associated cytoplasm, termed a DNA unit (Moore and Mozdziak, 2004). The term satellite cell (based on its location) refers to the myogenic precursor cells located within the basal lamina of the muscle fibre adjacent to the sarcolemma (Figure 1). Satellite cells are capable of entering the cell cycle and proliferating and either fusing into existing fibres or fusing with each other to form new fibres (Haley et al., 2003).

There are two mechanisms of muscle growth; hyperplasia (increase in fibre number) and hypertrophy (increase in fibre size). The chick emerges from the egg with a predetermined number of muscle cell fibres that does not alter during the life of the bird. Muscle growth post-hatch is the result of an increase in myofibre size and an increase in DNA content (Haley et al., 2003; Mozdziak et al., 2002a); the latter increased DNA content is provided by satellite cells (Hill et al., 2003). However, instances of excessive hypertrophy such as that found in fast growing strains of poultry lead to idiopathic myopathy and an increased susceptibility to stress induced myopathy as a result of the muscle growing beyond the available blood supply (Maltby et al., 2004; Mitchell and Sandercock, 2004).

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Figure 1. Diagrammatic representation of the location of satellite cells in the muscle cell fibre (McFarland, 1999).

Apoptosis (nucleic turnover) in this instance refers to the removal of the nuclei units from the cell and not the death of the entire cell. In some instances the mechanism of nuclei turnover can compete with muscle growth as a reduction in the number of DNA units available to the muscle cell would result in a decrease in muscle size throughout the life of the animal. Evidence suggests that there is some flexibility in DNA unit size, however once a critical level is reached, the superfluous myonuclei are destroyed (Mozdziajk et al., 2002b). Apoptosis of myonuclei in post hatch starved chicks has been attributed to irreversible reductions in muscle size (Mozdziajk et al., 2002b).

At hatch, skeletal muscle of the chick contains a high proportion of proliferating satellite cells that decrease rapidly towards the end of the growth cycle (Haley et al., 2000). This period of proliferation and differentiation has been reported to last only one week post hatch in broilers (Haley et al., 2003) where in mature animals, satellite cells are largely quiescent unless activated by myotrauma (muscle damage). The response to myotrauma results in regulated satellite cell populations migrating to the site of injury and depending on the extent of injury, either fuse to the existing myofibre or produce a new myofibre (Hawke and Garry, 2001). Thus any attempt to increase the number of muscle fibre cells would be best made during the embryonic and early post hatch stages of development.

The process of satellite cell activity and therefore muscle growth is influenced by several growth factors and other stimuli (Figure 2). Secretion of Insulin-like Growth Factor (IGF-I and IGF-II) by skeletal muscle has been determined to be particularly important in the proliferation and differentiation of satellite cells in vitro (Hawke and Garry, 2001). This work is supported by studies conducted by Chakravarthy et al. (2001) who reported that intramuscular administration of IGF-I resulted in enhanced satellite cell proliferation and increased muscle mass.
Figure 2. Factors that modulate satellite cell activity (Hawke and Garry, 2001).

III. MANIPULATION OF SATELLITE CELLS

a) Nutrition

The nutrition of the post-hatch chick has also been demonstrated to influence satellite cell activity. Work conducted by Halevy et al. (2000, 2003) has shown that starvation of the post-hatch chick for 48 h leads to a decrease in satellite cell proliferation resulting in decreased overall body and breast muscle weight when compared to chicks that were fed within 1 h post-hatch. During this adjustment period, the chick moves from being reliant on the yolk sac (lipid) as a source of nutrients to that of carbohydrate (starch) (Moran, 1995). During this adjustment phase, approximately 2-6% of hatchlings don’t survive, and many that do exhibit reduced growth, poor feed conversion, compromised immune status and poor meat yield (through apoptosis (programmed cell death); Uni and Ferket, 2004). If post-hatch starvation occurs, energy is diverted away from the proliferative satellite cells and redirected to mechanisms essential to survival (Moore and Mozdziak, 2004). As the broiler increases its body weight 50-fold from hatch until market age (42 days), the magnitude of this adjustment phase represents a greater proportion of the bird’s productive life and therefore importance to manage.

Limitations to satellite cell activity also reside in the underdeveloped gastrointestinal tract of the chick post hatch. Impaired development of the gastrointestinal tract has been associated with post hatch nutritional deficiencies, reduced satellite cell activity and decreased body weight at market age (Noy et al., 2001). Practical methods to overcome this
handicap are as yet to be fully elucidated however current research is targeting the administration of specific nutrients directly into the amnion of the of the late term embryo (in-ovo feeding) as well as the provision of a post-hatch feed source (early feeding) to stimulate gut development. The provision of a highly digestible early post hatch feed at the hatchery could be more attractive due to the relatively small amounts of feed consumed by the chick.

b) Chick Quality

Further compounding this situation of variable muscle mass at marketing age is the individual effect of the chick – chick quality. Chick quality is also highly variable and plays a significant role in the outcome of the hatching process (hatch %, body weight, mortalities) and inevitably influences the marketing weight/age of the bird (Boerjan, 2004). The vitality and performance of the chick is strongly influenced by breeder nutrition and immunocompetence, as well as the handling and storage of fertile eggs (York, 2004). As genetic potential for increased growth performance and production increases there will be further changes to requirements for embryo and early chick development. Chick quality and vitality is highly dependant on the incubator climate with increased mortalities and delayed development associated with temperatures greater and/or less than optimum (Boerjan, 2004).

c) Environmental

Further opportunities for satellite cell manipulation exist with temperature control both pre- and post-hatch. We hypothesise that these environmental effects may be linked with the nutrient profile of the egg, e.g. the ratio of egg yolk and albumen and fluctuations of specific nutrients in these two egg components. During the final stages of embryonic development, target tissues of the embryo become responsive to signals of heat and cold stress, with development and differentiation being inversely related to temperature. The need for different and highly specific incubation conditions was highlighted by studies that measured oxygen consumption of Ross 308 embryos during incubation; the Ross 308 embryos produce approximately 26% more metabolic heat than traditional meat producing strains (Boerjan, 2004). Exposure to constant high temperatures (> 39.5°C) inside the hatcher also results in stunted growth and poor chick vitality.

Halevy et al. (2001) investigated the effects of early age heat exposure and its affect on satellite cell proliferation and muscle development in chicks. In response to thermal conditioning (37 °C for 24h at 3 days of age) of the chick, increased IGF levels in the breast were observed; this was associated with an increased satellite cell proliferation and differentiation, indicating that IGF-I plays a central role in the immediate stimulation of satellite cell myogenic processes in response to heat exposure. Maximal improvements to satellite cell proliferation and differentiation were achieved when thermal conditioning was applied at day three post hatch and therefore may be applicable to industry as a means to improve breast yield. These results are contrary to the effects of heat exposure to broilers at 6 weeks of age, where exposure to similar temperatures leads to muscle damage (Yahav 1998), indicating that heat exposure at an early age results in an increase in growth factors, important to muscle hypertrophy.

The effects of light on the development and growth of chicks has also been shown to influence body weight and breast muscle weight. Studies by Rozenboim et al. (2004) investigated the effects of light on embryonic development, finding that stimulation with monochromatic green light during incubation enhanced development and growth in chicks, enhancing breast and body weight at market age whereas rearing under green light did not produce any noticeable affect. The mechanism for this is yet to be determined however it is
theorised that the use of green light during incubation increases the number of myoblasts therefore leading to increased muscle hypertrophy.

IV. CONCLUSION

The implications of this review indicate that muscle size and therefore meat yield may be enhanced by the stimulation of myogenic precursor satellite cells. Increased muscle fibre cell number would circumvent the increased myopathies of excessive hypertrophy whilst providing increases to meat yield. Methods such as, alternative breeder nutrition, in-ovo feeding, enhanced environmental control of incubation and early nutrition may provide practical methods to achieve this, however more research is required to determine the mechanisms of this phenomenon.

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REFERENCES